

MIGHT THE TIME BE RIPE TO RE-DEFINE THE CAUSE OF AIDS IN THE LIGHT OF EMERGING EVIDENCE?

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The assumption proposed by Gallo in 1984 that the cause of AIDS is infection with HIV was founded on the correlation between detection of antibodies to this virus and the onset of AIDS. This view became generally accepted, and today it is still the foundation stone of HIV-related measures for the prevention and treatment of AIDS. However, although unanimously rejected by AIDS researchers to date, Duesberg vehemently opposed this opinion, suggesting that AIDS is caused by drugs and malnutrition and that HIV is only a passenger pathogen (Duesberg, 1988; 1994; Lindermann, 1994). Earlier, it was reported that zinc deficiency was becoming a reality in the UK and the problem might be worse in developing countries, yet zinc deficiency and AIDS symptoms were similar and that micro-nutrient zinc had anti-viral, anti-bacterial and anti-cancer properties, for reasons that were only poorly understood then (Bryce-Smith, 1989).

More recently, the role of consumption of fumonisin-contaminated maize has been linked with increased risk of HIV transmission, while consumption of rice contaminated with aflatoxins has been associated with hepatocellular cancer (Williams *et al.*, 2010), suggesting that there may be other co-factors in the HIV and AIDS disease as earlier observed by Professor Duesberg. More than a decade earlier, a literature review paper suggested that the search for a cure for HIV and AIDS would remain extremely elusive, and for a long time to come, if the combined role of micro-nutrient zinc and the immune-suppressing dioxins was relegated to the periphery of biomedical research (Mbakaya and Wakori, 1997). Soon after, it was reported that zinc deficiency was responsible for the pre-mature switch from cellular (T-helper-1, Th-1) to humoral/antibody (T-helper-2) immunity (Sprietsma, 1999).

Furthermore, it has been observed that micro-nutrient zinc deficiency was a possible co-factor in the transmission and progression of HIV and AIDS in Kenya (Mbakaya *et al.*, 2004b), also indirectly supporting Duesberg's views. Lately, it has been argued that serum zinc levels and by extension cell-mediated immunity (Th-1) are physiologically lowered to produce antibodies (Th-2) in adapting to pathogens such as HIV (Mbakaya *et al.*, 2008a) and chemicals such as oral contraceptive agents (Mbakaya *et al.*, 2010c), perfectly elucidating the hitherto un-explained co-infection with malaria, HIV and AIDS,

TB, pneumonia and cancer since these diseases require cellular-immunity for effective prevention and control (Mbakaya *et al.*, 2006; Mbakaya , 2010; Mbakaya *et al.*, 2010f; Williams *et al.*, 2010). More recently, a study has shown that use of zinc microbicides impact a 100% protection in infection with SIM in monkeys (Kenney *et al.*, 2011), further anchoring the earlier views that zinc deficiency is a possible co-factor in HIV transmission and progression (Mbakaya *et al.*, 2004; Bryce-Smith, 1989). This further supports earlier views that zinc sufficiency may explain why monkeys are resistant to HIV-1 infection, suggesting that humans may be more deficient in zinc than monkeys due to lifestyle and dietary differences (Mbakaya, 2006).

An open labeled study in Kenya showed that by management of HIV and AIDS patients using micro-nutrients with enhanced antioxidant properties that included zinc, it was possible to reverse the signs and symptoms associated with HIV and AIDS, significantly reduce viral load and favourably increase the Th-1/Th-2 cytokine expression ratio 8-fold after 36 months of supplementation (Mbakaya *et al.*, 2004a) and support an earlier report that zinc deficiency and AIDS symptoms were similar (Bryce-Smith, 1989). Recently, it has been demonstrated in a randomised controlled study in the US that supplementation with zinc is effective in preventing immunological failure and diarrhoea in HIV and AIDS patients receiving HAART (Baum *et al.*, 2010). A study in South African in which children were supplemented with recommended daily allowance (RDA) zinc for 6 months observed significant reduction in the incidence of diarrhea, but had no effect on viral load (Bobat *et al.*, 2005). These findings are supported by those from another study in Kenya that showed that there was a correlation between low serum zinc, HIV seropositivity, enhanced antibody production and significant viral load reduction in adults on mega-dose multi-micro-nutrient supplementation in western Kenya, suggesting an evolutionary inter-play and a way forward in the fight against HIV and AIDS (Mbakaya *et al.*, 2008a). Therefore, it would appear that these recent studies consistently support the rejected views of Duesberg, to a large extent, when he refuted the HIV causes AIDS theory (Lindermann, 1994). However, to demonstrate how emotive and deeply entrenched the HIV causes AIDS views are among majority of scientists, one needs to look no further than the recent institution of a strict peer-review mechanism at the *Medical Hypotheses* journal, all because the editor and his editorial board (most of whom have since resigned) had, without peer-review and within their earlier editorial mandate of many years, published a purportedly controversial paper by Professor Peter Duesberg on the HIV causes AIDS theory.

Considering that the final whistle on the outcome of this heated scientific debate is yet to be blown, and none can claim the final laugh yet on this matter that is of major public health concern globally and that has been around and un-resolved for decades, this may be the time to throw more spanners in the works by suggesting that the need to redefine the cause of AIDS may long be overdue, given the emerging evidence.

Under the circumstances, we are scientifically persuaded to speculate and redefine that "AIDS is caused not only by HIV alone but by multiple factors, primarily including zinc

deficiency as a result of malnutrition and/or evolutionary dynamics that shift human immunological responses from predominantly Th-1 to Th-2 to adapt to/tackle offending pathogens (e.g. viruses, fungi, parasites, semen in the rectum/vagina, bacteria, etc.) and chemicals (pesticides, drugs, contraceptives, dioxins, fumonisins, aflatoxins, dibenzofurans, etc.)” (Mbakaya, 2011). Consequently, when this becomes acceptable as an alternative definition of the cause of AIDS, it will be impossible to envisage success against the scourge purely through awareness campaigns aimed at sexual behavior modification and HAART provision alone without shifting gear to wider efforts that endeavour to prevent populations, especially in sub-Saharan Africa, from widespread exposure to environmental, food-borne and lifestyle chemicals, pathogens and rampant malnutrition.

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